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A SURVEY OF THE ARTHROPOD VECTORS OF EQUINE ENCEPHALOMYELITIS AND ENCEPHALITIS*

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Equine encephalomyelitis is a disease that attacks horses, mules, donkeys, and other animals, causing a combined inflammation of the brain and spinal cord. The causative agent of this disease is a filterable virus with neurotropic properties (28). The three known strains of equine encephalomyelities virus, western, eastern, and Venezuelan, are serologically and immunologically distinct from one another.

Within the boundaries of North America occur both the western and eastern strains. The western strain of the virus, since its discovery in California during 1930–31, has been recovered in the states of Alabama, Arizona, Colorado, Idaho, Illinois, Iowa, Kansas, Kentucky, Michigan, Minnesota, Montana, Nebraska, Nevada, North Dakota, South Dakota, Texas, Utah and Washington. The eastern strain has been found in Alabama, Connecticut, Delaware, Florida, Georgia, Louisiana, Maryland, Massachusetts, Michigan, Missouri, New Jersey, North Carolina, South Carolina, Texas, and Virginia. As may be seen both strains have been found in Alabama, Michigan, and Texas. With the exception of Pennsylvania, Tennessee and West Virginia, from which epizootic encephalomyelitis had not been

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reported during the fifteen years preceding 1946, unidentified strains of the virus have been found in the remaining unnamed fourteen states (4, 6, 40, 72).

In the report of the Chief of the Bureau of Animal Industry for the year 1937 it was stated that the virus of equine encephalomyelitis had been definitely recovered in 22 states, and that during the summer and fall of 1937 more than 169,000 cases and approximately 40,000 deaths among horses had been reported (32, 68). In 1938, 184,662 cases among horses were recorded. The number of cases tabulated from 1935 through 1944 was 500,000, and Shahan and Giltner (72) have estimated that at least 1,000,000 cases occurred in the United States between 1930 and 1945. As the average mortality rate is about 30 per cent, it is possible therefore that since 1930 as many as 300,000 horses and mules have died of infectious equine encephalomyelitis.

In Canada, the western strain of the virus is predominant, with a few cases of the eastern strain occurring only in the region of Ontario. The eastern strain has also been identified in Mexico (72).

Equine encephalomyelitis is found in many of the Central and South American countries. Panama and Brazil have had cases of eastern strain virus within their borders. The western strain has been found in Argentina, where, according to Rosenbusch, "Outbreaks of equine encephalomyelitis occurred in horses over the whole agricultural zone of Argentina in the summer of 1919, another of less intensity in the centre and north of the country in 1933, and a third one in the eastern provinces in 1935–36. There were a few isolated cases in 1938–39" (41).

The Venezuelan strain of the virus has been found in Venezuela, Trinidad, Colombia, and Ecuador. In Trinidad, the Venezuelan strain had killed, as of October, 1943, approximately 70 horses and mules (37, 55, 72).

In recent years the equine encephalomyelitis disease has been observed also in Uruguay, Chile, Peru, and Cuba. During 1944 five samples of equine encephalomyelitis virus obtained from Cuba were typed and found to be all of the eastern type (37, 72).

Encephalitis, a disease of man in which the brain becomes inflamed, is caused by either the same virus strains that are responsible for equine encephalomyelitis or by other strains. The virus strains involved are serologically and immunologically distinct from one another.

These strains of human encephalitis virus are known as: St. Louis, Russian spring-summer, Russian autumn or Japanese "B," West Nile, and Semliki Forest. The St. Louis strain occurs in the United States and has been found in many of the states ranging from Massachusetts in the East to Washington on the Pacific Coast.

The virus of the tick-borne spring-summer encephalitis has been found in several parts of the Russian Union, including European districts not in the forest zone, and in Siberia (3, 21, 30). The virus of this disease is closely related to, or identical with, the virus of louping-ill of sheep in Scotland, which is also tick-borne (2). The virus of the autumn encephalitis has been found in the maritime district and is identical with the Japanese "B" virus.

The Japanese "B" virus was isolated about the year 1934, ten years after it had caused one of the most severe epidemics of encephalitis described. During the summer of 1924 there had occurred in Tokyo over 6,000 cases of encephalitis with 3,797 resultant deaths. This virus was named "B" to distinguish it from "type A," or the von Economo type which was responsible for many cases of encephalitis between the years 1918 and 1926, but which has since vanished (63).

The virus of West Nile encephalitis was first isolated in 1937 from the blood of a native woman in the West Nile district of Uganda, Africa (36, 73).

Smithburn and Haddow, while investigating the vectors of the yellow fever virus in the Semliki Forest, Western Uganda, Africa, isolated a neurotropic virus from mosquitoes of the *Aedes abnormalis* Theobald group. The virus has been named the Semliki Forest virus (74).

EQUINE ENCEPHALOMYELITIS

In 1933, R. A. Kelser reported the first successful transmission of the virus of equine encephalomyelitis (western strain) by an

insect vector. Female adults of *Aedes ægypti* L., fed on guinea pigs 48 to 72 hours after the inoculation of the latter with virus of equine encephalomyelitis, became infected. Sixteen to eighteen days after the infective meal the mosquitoes transmitted the disease to guinea pigs and to a horse on which they were fed. The infected animals died (17).

Under natural conditions, *Culex tarsalis* is the chief vector of western equine encephalomyelitis, as has been shown by the works of Hammon and his associates. They isolated strains of western equine encephalomyelitis virus from *C. tarsalis* collected in areas in which the disease was both epidemic and epizootic, and later this species transmitted the western virus in laboratory experiments (10–13, 61–62). *Culiseta inornata* has been proved to be a natural vector of western strain virus as it has been found naturally infected, and has transmitted the virus in laboratory tests (62). *Culex pipiens* and *Anopheles maculipennis freeborni* have been found naturally infected with western equine encephalomyelitis virus, but no experimental transmission has been demonstrated with them. Therefore, these two species are not generally considered to be vectors of the western strain.

To date, no arthropod vector has been found in the United States for eastern equine encephalomyelitis virus. A Japanese report of 1940 (given by Hammon and Reeves, Amer. Jour. of Public Health, Vol. 35, pp. 994–1004, 1945) claims transmission of eastern strain by *Culex pipiens* var. *pallens* and by *C. tritæniorhynchus*. *C. pipiens* var. *pallens* gave negative results with American workers.

Arthropod transmission of the viruses of encephalomyelitis and encephalitis has been summarized in Tables 1 and 2.

Relationship of Virus to Vectors.—Merrill *et al.* (25) found by laboratory experiments that in order for *Aedes ægypti* and *Aedes sollicitans* to become vectors of either western equine encephalomyelitis or eastern equine encephalomyelitis, they must be fed on infected animals at a time when the virus content of the blood is such that 0.0001 cc. or less will produce the disease when it is injected into a guinea pig. When these two mosquitoes fed on infected animals that had a lower virus content

in their blood, the virus was soon lost and the mosquitoes did not transmit the disease.

A period of 4–5 days must elapse after *A. ægypti* has fed on an adequately infected guinea pig or on a brain suspension containing the virus before it can transmit the virus of the western strain (26).

Aedes nigromaculis and *Aedes dorsalis*, fed on infected guinea pigs at intervals of from 12 to 72 hours after the latter had been injected with the virus of the western type, were allowed to bite healthy guinea pigs after intervals ranging from 3 to 24–25 days. Positive transmission was obtained with the mosquito *A. nigromaculis* when individuals had fed 18–66 hours after injection of the guinea pigs and had bitten healthy guinea pigs 4–10 days after their infecting meal. The greatest percentage of positive transmissions was obtained on the 6th, 7th, and 8th day. In tests using *A. dorsalis*, the positive results obtained were not very definite, but those that did occur were obtained with mosquitoes that had fed 18–42 hours after injection of the diseased guinea pigs and had bitten healthy animals 9–19 days after the infective meal (20, 24).

Merrill *et al.* (25) demonstrated that in both *A. ægypti* infected with western type and *A. sollicitans* infected with eastern type, the quantity of virus increased 1000–10,000 times within the mosquito.

Merrill and Ten Broeck (27) presented proof of the multiplication of equine encephalomyelitis virus within the mosquito vector by means of serial passage of the virus from mosquito to mosquito. The method used is quoted as follows:

> Thirty female *A. aegypti* infected five days previously by feeding on brain virus of the western strain of equine encephalomyelitis were suspended in 4 cc. salt solution plus 1 cc. normal horse serum. An equal amount of defibrinated horse blood was added and a pledgit of cotton in a Petri dish was moistened with the mixture. A small amount of sugar was sprinkled over the surface of the cotton and the Petri dish was placed in a cage containing female *A. aegypti* that had had no sugar solution for four days and no water for one day. Since the virus deteriorates rapidly when in contact with the air at room temperature, the Petri dish was replaced in an hour's time by one containing the mixture that had been kept in the refrigerator. After another hour this was removed, so that the mosquitoes that fed took up active virus. Those that did not feed were

eliminated by withholding water for 24 hours and sugar solution for 48 hours from the entire lot. The infected mosquitoes were kept in cages at a room temperature of 24–28° C.

At six to seven day intervals from 25–30 mosquitoes from the last feeding have been suspended and fed to starved females as outlined above. At each transfer virus has been demonstrated in the suspension of crushed mosquitoes by guinea pig inoculations and in many instances dilutions as high as 10^{-5} have proven infectious. Control inoculations of three kinds into guinea pigs have all been negative: a suspension of mosquitoes from our healthy stock; the horse serum and saline used; and a boiled suspension of infected mosquitoes. Since the virus has now been passed in series through ten lots of mosquitoes and since the dilution at each transfer is at least 1:100 we must conclude that multiplication has taken place.

No difference has been demonstrated between the mosquito passage virus and the original strain. Its serological characters are unchanged, the virulence has been modified little if at all, and it passes Berkefeld N filters readily. Mosquitoes infected with the passage strain readily infect guinea pigs by biting.

Merrill and Ten Broeck (27), in the course of their investigations on the multiplication of western equine encephalomyelitis virus in mosquitoes, concluded that the virus appeared to be generally distributed in the body of the mosquitoes. This was determined by inoculating guinea pigs with suspensions of legs removed from uncrushed infected insects, as well as with suspensions of the body fluid, heads, thoraces, and abdomens.

In 1934, Merrill *et al.* determined that the eastern strain virus appeared to persist in at least some of the vectors as long as the latter lived. *A. sollicitans* transmitted the virus of eastern equine encephalomyelitis 33 days after the infective meal. Females of *A. ægypti* were able to transmit the western type after 63 days, and 93 days after the infective meal the virus was shown to be still present within the mosquitoes, although they were not transmitting it at the time (25).

Davis (4) found that the longest time after the infective meal at which *A. ægypti* transmitted the virus of equine encephalomyelitis was 41 days.

Merrill and Ten Broeck (26) found in the course of their investigations with *A. ægypti* as vector of western equine encephalomyelitis that the virus strain could not be demonstrated in eggs from females known to be infected or in larvæ,

pupæ, and adults reared from such eggs. The larvæ did not take up the virus when it was added to their rearing water.

In laboratory experiments, Syverton and Berry (48, 49) demonstrated that *Dermacentor andersoni*, a wood tick vector of western equine encephalomyelitis could, in its early stages, acquire the virus, carry it to later stages in its life cycle and also to its progeny. No naturally infected ticks have been found.

Merrill and Ten Broeck (26) showed by laboratory experiments that males of *A. œgypti* could become infected with the virus of western equine encephalomyelitis by feeding on a suspension of virus containing brain tissue and horse blood. Eighteen to twenty-five days after the mosquitœs fed, virus was demonstrated in two suspensions of fifteen and twelve male mosquitoes respectively. However, the remaining infected males did not transmit the virus to normal females, nor did they transmit it from infected to normal females by coition.

It was found that the virus of western equine encephalomyelitis was apparently not injurious to the vector, *A. œgypti*, for the mortality of caged infected mosquitoes was no higher than that of caged uninfected mosquitoes (25).

Reservoirs of Eastern Equine Encephalomyelitis Virus.—In 1935 Ten Broeck *et al.*, (77) favored the view that equine encephalomyelitis of the eastern type was insect-borne, and mentioned the possibility that the disease was not primarily an infection of horses but that it was transmitted to them from birds. Again, in 1938, Ten Broeck (78) after finding neutralizing antibodies in the blood of chickens and turkeys injected with equine encephalomyelitis virus, concluded that domestic and probably wild birds may be infected with equine encephalomyelitis, and that birds may play a part in the transmission of the disease.

In 1939, Van Roekel and Clarke reported the isolation of eastern type equine encephalomyelitis in ring necked pheasants. The pheasants had been obtained from New Jersey during the 1938 outbreak of encephalomyelitis in New Monmouth, Monmouth County (52, 80).

During 1938, Tyzzer *et al.*, (79) found the eastern type equine

encephalomyelitis virus in three ring-necked pheasants. At the same time Fothergill and Dingle (58) recovered the eastern type from the brain of a pigeon which had spontaneously contracted the disease in an area where equine encephalomyelitis was prevalent among horses. Sellards *et al.* recovered encephalomyelitis virus (eastern type) from dying pheasants in Connecticut (71).

Following laboratory experiments, Davis (4) maintained that mourning doves (*Zenaidura macroura*), redwings (*Agelaius phœnicens*), cowbirds (*Molothrus ater*) and grackles (*Quiscolus quiscula*), all migratory species often seen in close association with livestock, might serve as reservoirs from which mosquitoes become infected. The above listed birds were susceptible to the virus, and for a short time following inoculation, Aedes mosquitoes recovered the virus from the blood and transmitted it.

Beaudette reported the presence of eastern type equine encephalomyelitis in pheasants in New Jersey during 1939 (three distinct outbreaks), 1940, 1943, and 1944 (two outbreaks) (52, 53, 54).

Reservoirs of Western Equine Encephalomyelitis Virus.—In 1941 Hammon *et al.* (9) found that in the annual epidemics of western equine encephalomyelitis and St. Louis encephalitis in horses and man in the Yakima Valley, Washington, the antibodies of these two viruses could be demonstrated in fowls, ducks, geese, pigeons, turkeys, other birds, cows, dogs, goats, horses, pigs, sheep, and rodents by means of mouse protection tests. Apparently the antibodies in many of the animals listed above are the result of specific infection, either mild or inapparent. The principal foci of infection could be the many barnyards and fowl runs in small towns, rural and suburban areas. The domestic species of animals produced a higher percentage of positives than did the wild species.

Cox *et al.* reported in 1941 the finding of western equine encephalomyelitis virus from a naturally infected prairie chicken. As of 1941, the western strain of virus had been isolated from the brain tissues of eight human cases, three horses, one prairie chicken and one deer (57).

Hammon *et al.* (13) found that in the 1943 survey made in

eastern Nebraska, in which neutralization tests were conducted on the sera of 91 mammals and birds, positive results were obtained with pheasants and horses for St. Louis encephalitis and with cows for western equine encephalomyelitis. The investigators concluded that as all results with fowls, rabbits, and pheasants were negative for western equine encephalomyelitis the virus may be adapted to some other host in that locality.

In 1940 Howitt (67) reported that no western equine encephalomyelitis virus was isolated from the brains of 43 wild animals and birds, representing 14 different species taken in the endemic area of the Central California Valley regions as well as from certain coastal districts. Included among those tested for the virus were Gambel sparrows, Kangaroo rat, harvest mouse, pocket gopher, mourning doves, painted finch, wood rat, cottontail rabbit, mallard ducks, pintail duck, and the ring necked pheasant.

Reeves *et al.* have reported the recovery of western strain virus from wild bird mites, (*Liponyssus sylviarum*), in Kern County, California (70). The mites were taken from the nests of two wild birds, and in laboratory tests were shown to harbor the virus.

A variety of birds have been found to be susceptible to infection by either the western or eastern strain of the virus in laboratory tests. Giltner and Shahan (59) made the first successful experimental inoculation of equine encephalomyelitis (western strain) into birds. The following workers have contributed additional information on experimental infection in avian hosts: Beaudette (52), Gwatkin and Moynihan (60), Howitt (67), Sellards *et al.* (71), Syverton and Berry (76), Ten Broeck (78)), Tyzzer *et al.* (79), and Van Roekel and Clarke (80).

Winter Reservoir of Equine Encephalomyelitis Virus.— Hammon and Reeves (64) indicate that the problem of finding a true reservoir (or winter carry-over) of western equine encephalomyelitis virus remains unsolved. The relationship between the vectors and the summer reservoirs of the virus has been well established, but nothing is known concerning its winter

reservoirs. Hibernating *C. tarsalis* adults have been tested for the presence of virus and found negative. No transovarian infection has been found in *C. tarsalis*, and no persistent latent infection has been found in mammals.

The true reservior of eastern equine encephalomyelitis is also completely unknown.

Encephalitis in Horses.—Cox et al. have presented data demonstrating that horses are susceptible to the virus of St. Louis encephalitis. Typical clinical symptoms were produced in experimental infections. Horses that showed antibodies for western equine encephalomyelitis in high titer were susceptible to St. Louis encephalitis virus, while horses demonstrating St. Louis encephalitis antibodies as the result of natural infection were apparently resistant to subsequent infection by St. Louis encephalitis virus (56).

ENCEPHALITIS

Several years after Kelser had demonstrated the transmission of equine encephalomyelitis by *Aedes ægypti*, definite proof was obtained that mosquitoes were involved in the transmission of human encephalitis. Epidemics of encephalitis had occurred in Yakima Valley, Washington, in 1939 and 1940 but it was not until 1941 when many cases of encephalitis again occurred in Yakima Valley that routine collections of insects were made in the area, and subsequent work resulted in the isolation of the St. Louis strain from *Culex tarsalis*. Experimental transmission of the virus to laboratory animals by the bite of this species was obtained at a later date. (Virus of western equine encephalomyelitis was also isolated from some of the specimens of *C. tarsalis*.) (10, 61).

Culex pipiens and *Aedes dorsalis* have been found naturally infected with virus of St. Louis encephalitis, and transmission has been demonstrated in the laboratory for *C. pipiens*. Therefore, *C. tarsalis* and *C. pipiens* are now considered established vectors (12, 64). Hammon and Reeves have demonstrated experimental transmission by *Aedes lateralis, A. tæniorhynchus, A. vexans, A. nigromaculis, Culiseta inornata, Culiseta incidens* and *Culex coronator*. Japanese workers have reported the transmission of St. Louis virus by *C. pipiens* var. *pallens, C.*

tritæniorhynchus and *Aedes albopictus* (given by Hammon and Reeves, Amer. Jour. of Public Health, Vol. 35, pp. 994–1004, 1945).

Previously, in 1939, E. N. Pavlovskii (46) asserted that three ticks, *Ixodes persulcatus, Dermacentor silvarum,* and *Hæmophysalis concinna,* had been found naturally infected with virus of Russian spring-summer (or taiga) encephalitis in localities where cases of the disease had occurred.

Smith *et al.* (44) and Sulkin (45) reported in 1945 the presence of both the St. Louis encephalitis virus and the western equine encephalomyelitis virus in the chicken mite, *Dermanyssus gallinæ.* The mites were collected from chicken houses in areas where outbreaks of these diseases had occurred.

Infection of Man with Virus of Equine Encephalomyelitis.—Webster and Wright reported in 1938 the recovery of eastern equine encephalomyelitis virus from fatal human cases of encephalitis in the state of Massachusetts. By doing so they confirmed the findings of Fothergill *et al.,* who in 1938 reported the isolation of the eastern strain virus from a human case (51).

During the same year Howitt reported the recovery of the western equine encephalomyelitis virus from the brain of a child (65, 66).

In 1943 the first proved case of natural infection of man with Venezuelen virus was recorded, a fatal case occurring in Trinidad. A virus strain isolated from the brain tissue of the fatal human case and five strains from donkeys, mules, and horses all produced typical symptoms of equine encephalomyelitis in laboratory animals. Injections of these strains into guinea pigs immunized against the western or eastern strains of equine encephalomyelitis resulted in the deaths of the animals. However, the injections failed to infect guinea pigs immunized aganst the Venezuelan strain of equine encephalomyelitis (37).

Relationship of Virus to Vectors.—Parker reported in 1942 that in the case of *Ornithodoros moubata,* Murr., tick harborer of Russian spring-summer encephalitis virus, the interval between the ingesting of the blood of the infected animal and the recovery of the virus by injection was forty days (33).

Experiments with suspensions made from the various organs

of *Hæmophysalis concinna*, tick vector of Russian spring-summer encephalitis, showed that the virus circulates in the body of the tick. The virus concentrates in the salivary glands and is probably transmitted to man through the saliva (46).

Several specimens of *Ixodes persulcatus*, Schulze, a tick vector of Russian spring-summer encephalitis virus, were allowed to feed on mice infected with a large dose of the virus. Tests showed that the ticks conserved the virus within their bodies for 60 days (46).

In 1945 Smith *et al.* (44), working on the transmission of St. Louis encephalitis virus by the chicken mite *Dermanyssus gallinæ*, demonstrated that mites harboring the virus could transmit it through the egg and larva to the first stage nymph. Mites infected in nature have retained the virus after 5 months of propagation in the laboratory.

Experiments with nymphs and adults of the tick *Dermacentor silvarum* (a vector of Russian spring-summer encephalitis) that had fed in the larval stage on infected mice transmitted the virus by feeding on laboratory animals. The virus was also present in larvæ of *D. silvarum* and *I. persulcatus* that were the progeny of naturally infected females. Injection of a suspension of larvæ of *I. persulcatus* that hatched from eggs laid by females collected in an endemic area produced infection in mice (46).

During 1939, it was shown in Japan that virus of Japanese epidemic or summer encephalitis was present in eggs deposited by artificially infected *Culex pipiens* var. *pallens*, a vector of the virus disease. The virus was also demonstrated in the first instar larvæ hatching from such eggs. In addition, twelve mice became infected out of 511 bitten by females of this species that had developed in the laboratory from larvæ and pupæ taken in nature (31).

No infection was found in the immature stages of *C. pipiens* L. and *C. tritæniorhynchus*, vectors of Russian autumn encephalitis (35).

Reservoirs of St. Louis Encephalitis Virus.—As mentioned in the discussion of the virus reservoirs of western equine encephalomyelitis, Hammon *et al.* (9) demonstrated antibodies of St. Louis encephalitis in fowls, ducks, geese, pigeons, turkeys, birds, cows, dogs, goats, horses, pigs, sheep, and rodents. Later, in

1943, Hammon *et al.* (12) demonstrated that *Culex tarsalis,* a vector of St. Louis encephalitis and western equine encephalomyelitis, fed readily on fowls. St. Louis virus was transmitted by it from fowls and from ducks to fowls in laboratory tests.

Philip *et al.* reported in 1941 that the presence of antibodies for St. Louis encephalitis virus had been demonstrated in the serum of man and horses. The investigators suggested that the virus existed as a natural infection in horses, thereby contributing to the summer encephalitis epidemics in both man and horses (69).

Hammon *et al.* (13) reported that in the 1943 eastern Nebraska survey in which neutralization tests were conducted on the sera of 91 mammals and birds, positive results were obtained with pheasants and horses for St. Louis virus.

Reservoirs of Russian Spring-Summer Encephalitis Virus.—Pavlovskii (34) found a mole, a hedgehog, and a vole naturally infected with the virus of Russian spring-summer encephalitis. The hedgehog and vole are known hosts of *I. persulcatus* and *H. concinna,* two tick vectors of the virus. Pavlovskii maintained that it is probable that an inapparent infection in man and animals, followed by immunity, is of common occurrence, as antibodies were present in the sera of cows, horses, and healthy persons in an endemic locality.

Reservoirs of Japanese Encephalitis Virus.—In Japan, dogs are often inapparent reservoirs for the virus of Japanese epidemic encephalitis, as shown by the presence of antibodies. (31).

Winter Reservoirs of Encephalitis Virus.—The true reservoir of St. Louis virus during periods or seasons when it is apparently not present is unknown. The two chief vectors, *C. pipiens* and *C. tarsalis,* do not demonstrate transovarian passage of the virus, nor do the hibernating adults of these two species appear to be infected. Experimentally infected dog ticks (*Dermacentor variabilis*) are capable of transmitting the infection, and transovarian infection can occur, but no naturally infected tick has been found (64). Chicken mites (*Dermanyssus gallinæ*), which have been found naturally infected with St. Louis virus, can transmit the virus transovarially with resultant infected progeny. But the mites cannot transmit the virus by bite, thus eliminating themselves as potential reserviors (44, 64).

The tables summarize the following data:

Table 1. Mosquito transmission of equine encephalomyelitis and of encephalitis.

Table 2. Arthropod (other than mosquito) transmission of equine encephalomyelitis and of encephalitis.

Table 3. Mosquitoes that failed to transmit equine encephalomyelitis in laboratory experiments.

Table 4. Arthropods (other than mosquitoes) that failed to transmit equine encephalomyelitis in laboratory experiments.

Table 5. Animals demonstrating antibodies of equine encephalomyelitis and encephalitis.

Table 6. Animals found infected in field with either equine encephalomyelitis or encephalitis.

TABLE 1

MOSQUITO TRANSMISSION OF EQUINE ENCEPHALOMYELITIS AND OF ENCEPHALUS

Vector	Virus strains transmitted								Virus demonstrated in	
	Equine encephalomyelitis			Encephalitis						
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)	Field	Lab.
1. *Aedes ægypti*	x	x	x							x (a) (b) (c)
2. *Aedes sollicitans*	x	x								x (a) (b)
3. *Aedes cantator*	x	x								x (a) (b)
4. *Aedes nigromaculis*	x			x						x (a) (d)
5. *Aedes dorsalis*	x			x					x (d)	x (a)
6. *Aedes albopictus*	x		x	x		x	x		x (a) (g)	x (a) (c) (d) (g)
7. *Aedes taeniorhynchus*	x			x						x (a) (d)
8. *Aedes vexans*	x	x		x						x (a) (b) (d)
9. *Aedes triseriatus*		x								x
10. *Aedes atropalpus*		x								x
11. *Aedes geniculatus*			x							x
12. *Aedes lateralis*				x						x
13. *Aedes togoi*						x				x
14. *Aedes japonicus*						x				x
15. *Aedes abnormalis* Theobald group								x		x
16. *Culex stigmatosoma*	x								x	
17. *Culex pipiens*	x			x	x				x (d) (e)	x (a) (d)
18. *Culex tarsalis*	x			x					x (a) (d)	x (a) (d)
19. *Culex pipiens* var. *pallens*	x	x		x		x			x (f)	x (a) (b) (d)
20. *Culex tritæniorhynchus*		x		x	x	x			x (e) (f)	x (b) (d) (e) (f)
21. *Culex coronator*				x						x
22. *Culiseta inornata*	x								x	
23. *Culiseta incidens*	x			x						x (a) (d)
24. *Mansonia titillans* complex			x						x	
25. *Anopheles maculipennis freeborni*	x								x	

Note: Lower case letters refer to virus strain: (a) Western, (b) Eastern, (c) Venezuelan, (d) St. Louis, (e) Russian Autumn, (f) Japanese, (g) West Nile, (h) Semliki Forest.

TABLE 1. (*Continued*)

Vector	Virus transmitted experimentally		Reference
	From	To	
1. *Aedes ægypti*	(a) Guinea pigs	Guinea pigs & horse	(a) 18, 25, 26, 40; (b) 4; (c) 42
	(b) Birds & mammals	Birds & mammals	
	(c) Guinea pigs	Guinea pigs	
2. *Aedes sollicitans*	(a) Guinea pigs	Guinea pigs	(a) 25, 40; (b) 4, 25
	(b) Mouse & sparrow	Mouse & sparrow	
3. *Aedes cantator*	Mouse	Mouse	(a) 40; (b) 4, 25
	Guinea pigs	Guinea pigs	
4. *Aedes nigromaculis*	(a) *A. nigromaculis*	Lab. animals & horses	(a) 23, 24, 40; (d) 64
	(a) Guinea pigs	Guinea pigs	
5. *Aedes dorsalis*	(a) *A. dorsalis*	Lab. animals & horses	(a) 20, 23, 24, 40; (d) 64
	(a) Guinea pigs	Guinea pigs	
6. *Aedes albopictus*	(a) (c) Guinea pigs	Guinea pigs	(a) 40, 43; (c) 42; (d) 64; (g) 36
	(g) Hamsters	Hamsters	
7. *Aedes tæniorhynchus*	(a) Guinea pigs	Guinea pigs	(a) 18, 40; (d) 12
	(d) Blood virus suspension	Fowls	
8. *Aedes vexans*	(a) *A. vexans*	Lab. animals & horses	
	(b) Birds & mammals	Birds & mammals	
	(d) Blood virus suspension	Fowls	(a) 40; (b) 4; (d) 12
9. *Aedes triseriatus*	Birds & mammals	Birds & mammals	4
10. *Aedes atropalus*	Birds & mammals	Birds & mammals	4

Note: Lower case letters refer to virus strain. Numerals refer to reference list.

TABLE 1. (*Continued*)

Vector	Virus transmitted experimentally		Reference
	From	To	
11. *Aedes geniculatus*	Guinea pigs	Guinea pigs	42
12. *Aedes lateralis*	Blood virus suspension	Fowls	12
13. *Aedes togoi*	*A. togoi*	Mice & monkeys	64
14. *Aedes japonicus*	*A. japonicus*	Mice & monkeys	64
15. *Aedes abnormalis* Theobald group	Inoculations	Mice, guinea pigs, rabbits rhesus & red tailed monkeys	74
16. *Culex stigmatosoma*			64
17. *Culex pipiens*	(d) Virus suspension	Doves & mice	(a)(d) 38, 39, 10; (a) 5; (e) 35; (d) 12
18. *Culex tarsalis*	(a) Duck & guinea pig (d) Fowls & ducks	Fowls Fowls & ducks	(a)(d) 10, 11, 12, 39; (a) 13
19. *Culex pipiens* var. *pallens*	*C. pipiens* var. *pallens*	Mice	(a)(b)(d) 64; (f) 31
20. *Culex tritæniorhynchus*	(e) (f) *C. tritæniorhynchus*	Mice	(b)(d) 64; (e) 35; (f) 31
21 *Culex coranator*	Blood virus suspension	Fowls	12
22. *Culiseta inornata*			39, 62
23. *Culiseta incidens*	(a)(d) Blood virus suspension	Fowls	(a)(d) 12
24. *Mansonia titillans complex*			7
25. *Anopheles maculipennis freeborni*			39, 62

TABLE 2

ARTHROPOD (OTHER THAN MOSQUITO) TRANSMISSION OF EQUINE ENCEPHALOMYELITIS AND ENCEPHALITIS

Vector	Virus strains transmitted				Virus isolated in	
	(a)	(b)	(c)	(d)	Field	Lab.
1. *Dermacentor andersoni*	x					x
2. *Dermacentor marginatus*				x	x	x
3. *Dermacentor variabilis*			x			x
4. *Dermacentor silvarum*				x	x	x
5. *Dermanyssus gallinæ*	x		x		x (a) (c)	x (a) (c)
6. *Triatoma sanguisuga*	x				x	x
7. *Triatoma infestans*		x				x
8. *Ixodes persulcatus*				x	x	x
9. *Hæmaphysalis concinna*				x	x	x
10. *Ornithodoros moubata*				x		x
11. *Liponyssus sylviarum*	x				x	x

Note: Lower case letters refer to virus strain: (a) Western, (b) Venezuelan, (c) St. Louis, (d) Russian Spring-Summer.

TABLE 2. (*Continued*)

Vector	Virus transmitted experimentally		Host in field	Reference
	From	To		
1. *Dermacentor andersoni*	Guinea pigs	Gophers	Gophers (*Citellus richardsoni*)	47, 48, 49
2. *Dermacentor marginatus*	Rabbits	Rabbits	Horses	16
3. *Dermacentor variabilis*	Mice	Mice		1
4. *Dermacentor silvarum*	Mice	Mice	Man, domestic animals, rodents, thrush	34, 46
5. *Dermanyssus gallinæ*	(a) Triturated mites	Mice & guinea pigs	Fowls	(a) 45; (c) 44
	(c) " "	Mice		
6. *Triatoma sanguisuga*	Guinea pigs	Guinea pigs		8, 19
7. *Triatoma infestans*	Guinea pigs	Guinea pigs		22
8. *Ixodes persulcatus*	Mice	Mice	Man, domestic animals, rodents, thrush	3, 46
9. *Hæmaphysalis concinna*	Mice	Mice	Man, domestic animals, rodents, thrush	46
10. *Ornithodoros moubata*				33
11. *Liponyssus sylviarum*	Mice	Mice, guinea pigs, hamsters	Wild Birds	70

TABLE 3

MOSQUITOES THAT FAILED TO TRANSMIT EQUINE ENCEPHALOMYELITIS IN LABORATORY EXPERIMENTS

Mosquito	Strain of virus used in attempted transmission	Attempted virus transmission		Reference
		From	To	
1. *Mansonia perturbans*	Eastern	Birds & mammals	Birds & mammals	4
2. *Anopheles punctipennis*	Eastern	" "	" "	4
3. *Anopheles malculipennis* var. *atroparvus*	Venezuelan	Guinea pig	Guinea pig	42
4. *Anopheles quadrimaculatus*	Eastern and Western	"	"	25
5. *Culex salinarius*	Eastern	Birds & mammals	Birds & mammals	4
6. *Culex pipiens*	Eastern	" "	" "	4
7. *Aedes tæniorhynchus*	Eastern	Guinea pig	Guinea pig	18

Note: Numerals refer to reference list.

TABLE 4

ARTHROPODS (OTHER THAN MOSQUITOES) THAT FAILED TO TRANSMIT EQUINE ENCEPHALOMYELITIS IN LABORATORY EXPERIMENTS

Arthropod	Strain of virus used in attempted transmission	Reference
1. *Siphona irritans* L. (horn fly)	Western	15
2. *Tabanus punctifer* (horse fly)	"	15
Tabanus sp.	"	60
3. *Stomoxys calcitrans* (stable fly)	"	32
Stomoxys sp.	"	60
4. *Dermacentor variabilis* (tick)	"	8
5. *Cimex lectularius* (bedbug)	"	13
6. *Zelus audax* (assassin bug)	"	60
7. *Sinea diadema* (assassin bug)	"	60
8. *Chrysops* sp. (deer fly)	"	60
9. (Black crickets*)	"	60
10. (Grasshoppers*)	"	60
11. *Simulium vittatum* (black fly)	" (results inconclusive)	20

Note: Numerals refer to reference list.

* Scientific name not given in reference.

TABLE 5

ANIMALS DEMONSTRATING ANTIBODIES OF EQUINE ENCEPHALOMYELITIS AND ENCEPHALITIS

Animal	Virus strain antibodies			Reference
	Equine encephalomyelitis	Encephalitis		
	(a) Western	(b) St. Louis	(c) Others	
1. Fowls	x	x		(a) 9, 61, 62; (b) 9, 62
2. Ducks	x	x		(a) 9, 61; (b) 9
3. Geese	x	x		(a)(b) 9
4. Pigeons	x	x		(a)(b) 9
5. Turkeys	x	x		(a)(b) 9
6. Cows*	x	x	Russian Spring-Summer	(a)(b) 9; (a) 13; (c) 34
7. Dogs	x	x	Japanese	(a)(b) 9; (c) 31
8. Goats	x	x		(a)(b) 9
9. Horses*	x	x	Russian Spring-Summer	(a) 9; (b) 13, 56, 69; (c) 34
10. Pigs	x	x		(a)(b) 9
11. Sheep	x	x		(a)(b) 9
12. Rodents	x	x		(a)(b) 9
13. Pheasants	x	x	Eastern	(a) 61; (b) 13; (c) 52, 53
14. Man	x	x	(1) Russian Spring-Summer (2) Semliki Forest	(a) 61; (b) 61, 62, 69; (c_1) 34; (c_2) 75
15. Primates (other than man)			Semliki Forest	(c) 75

Note: Lower case letters refer to virus strain. Numerals refer to reference list.

* Doubtful for Eastern Equine Encephalomyelitis (Ref. #13).

TABLE 6

ANIMALS FOUND INFECTED IN FIELD WITH EITHER EQUINE ENCEPHALOMYELITIS OR ENCEPHALITIS

Animal	Virus strain								Reference
	Equine encephalomyelitis			Encephalitis					
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)	
1. Gopher	x								47
2. Pheasant		x							4, 52, 53, 54, 79
3. Pigeon		x							4, 58
4. Syrian hamster	x	x					x		50
5. Rodent					x				46
6. Thrush					x				46
7. Horse	x	x	x		x				(a) 6, 32, 40, 57; (b) 6, 25, 32; (c) 37, 41; (e) 16
8. Cattle					x				46
9. Mule			x						37
10. Mole					x				34
11. Vole					x				34
12. Hedgehog					x				34
13. Man	x	x	x	x	x	x	x	x	(a) 9, 40, 57; (b) 4; (c) 37; (d) 9, 14; (e) 3, 29, 46; (f) 31; (g) 36, 73; (h) 75
14. Donkey			x						37
15. Prairie chicken	x								57
16. Deer	x								57

Note: Lower case letters refer to virus strain: (a) Western, (b) Eastern, (c) Venezuelan, (d) St. Louis, (e) Russian Spring-Summer, (f) Japanese, (g) West Nile, (h) Semliki Forest. Numerals refer to reference list.

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